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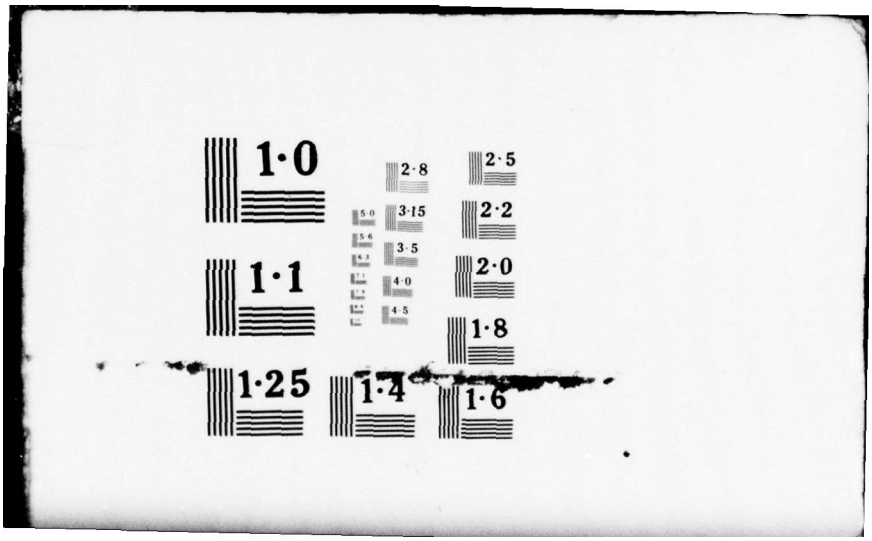
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LEVEL II

KNEE FLAIL DESIGN LIMITS: BACKGROUND, EXPERIMENTATION AND DESIGN CRITERIA

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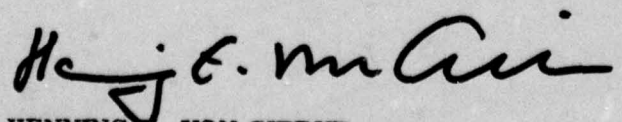

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A major problem experienced during ejection of crewmembers from disabled aircraft is flailing injury to the knee caused by the large aerodynamic forces. The majority of injuries that occur are tears of the collagenous tissues, ligaments and capsular structures, which hold the tibia and femur together at the knee. The design of protective systems capable of preventing these injuries requires knowledge of the mechanical properties of knee ligaments, and the

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factors which can effect the experimental results. This report combines new experimental data on the tensile strength of human anterior cruciate ligaments and torsional strength of the intact human knee with data on the strength of Rhesus monkey knee ligaments. The information is organized and presented so as to provide a background for relating mechanical property studies of ligaments and tendons to the establishment of design criteria for protective systems. In the last two chapters, the torsional strength data obtained under the current contract are presented and design criteria for the prevention of torsional injuries are developed. The effects of age and species on the tensile strength of the anterior cruciate ligament which were determined in the current contract are presented in greater detail in a separate Air Force technical report.

SUMMARY

The composition, structure, and mechanical properties of knee ligaments have been presented. Factors which affect the measurement of mechanical properties were reviewed. Storage of ligaments by freezing at -15°C for up to three weeks was found to have minimal effect on mechanical properties. The rate at which bone-ligament-bone units are tested affects both their strength and major failure mechanism. Bone avulsion fractures predominate at slow elongation rates while ligamentous tears predominate at high rates. The rate effects appear to act through the boney portion of the ligament insertion and not upon the ligament itself. In addition to the variables of storage method and test rate, it was found that species, donor age, and antemortem disuse and disease states can significantly affect both the mechanical properties and failure mechanisms.

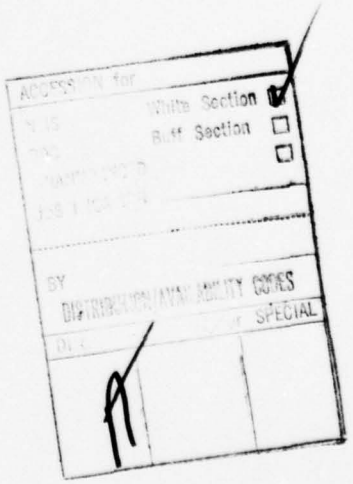
The ligamentous restraints, and the torsional failure limits of the human knee were presented. The data were then used to establish design criteria for the prevention of failing injuries during seat ejection. The limits were based upon the onset of failure as determined by the linear load point. The linear load point for soft tissues is analogous to the yield force of a metal. A safety factor of 1.5 was used in calculating the design limits.

The proposed limits are not recommended as operational design criterial because of unverified assumptions employed in their development. Future work should be directed towards verification of these assumptions. Specific studies should include:

Relating injury to the blood supply of ligaments to the amount of ligament elongation. This study would test our assumption that adequate nutrition was available for healing after sustaining an injury.

Comparison of the anthropometric characteristics of the Air Force pilots to be protected with those of the knee donors.

Studies of the rate of tissue healing following injury to properly establish treatment methods for injured crew members.



PREFACE

This work was performed under USAF Contract F33615-76-C-0511, entitled "Research on Limb Flail Design Limits." The contract was technically monitored by Captain Michael Higgins, Biomechanical Protection Branch, Biodynamics and Bioengineering Division, Aerospace Medical Research Laboratory, Wright-Patterson Air Force Base, Ohio. We wish to thank Captain Higgins for the continued support, interest, and direction he provided during the contract period. We also wish to thank Dr. Henning E. von Gierke, Mr. James W. Brinkley, and Dr. Leon Kazarian for their support and assistance on this and prior studies which provided the foundation upon which this effort was based.

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THE PROBLEM

In those instances when a manned aircraft becomes disabled, so that it may no longer be flown, safety requires separation of the crew members from the aircraft prior to its impact. The separation may occur by either bailing out, seat-ejection, or the ejection of an encapsulated crew station containing one or more crew members. When separation occurs by seat ejection, the crew member is subjected to a high velocity air stream which causes large drag, sideward and lift forces on his extremities. These aerodynamic forces can cause flailing of the extremities with subsequent injury, particularly of the soft tissues around the joints. The repeated occurrence of flailing joint injuries has demonstrated the need for improved ejection seat design capable of preventing the limb flailing and the resulting joint injuries. The development of flail resistant ejection seats requires that design limits be established for the allowable displacements and forces which the extremity joint structures may be subjected to. Establishment of the design limits requires two major steps. The first step is the specification of the allowable injury level, both the extent of the injury and frequency of occurrences. The second step is the determination of the mechanical limits upon joint displacements and forces which correspond to the allowable injury level within the population of Air Force pilots.

INJURY CRITERIA

In specifying the allowable injury to the ligamentous soft tissues it is important to realize that injury is a spectrum ranging from microtrauma, which occurs during normal daily activity, at one extreme to complete disruption with loss of load bearing ability at the other extreme. Clearly, specification of no injury to the ligamentous structures would be impossible to obtain and illogical from the standpoint of ligament function. Instead, what is desired is to limit the degree of injury to a level which may be repaired by normal metabolic tissue processes without producing significant permanent degradation in the functional capacity of the tissues. The problem of specifying injury levels is further complicated by the large amount of variability in biological systems. The forces and mechanical displacement which will produce a slight to moderate injury in one individual may produce a more severe injury with complete disruption in another individual. If a maximum injury level is specified which cannot be exceeded for any individuals, then the design limits must be based on the projected strength of the weakest ligaments in the population to be protected. An alternate approach is to limit the frequency of occurrence of a specified injury level. This, however, requires knowledge of the variation of ligament strength within the group to be protected, as well as the mechanical limits which correspond to the specified injury level.

MECHANICAL LIMITS

Determination of the allowable mechanical limits must be accomplished experimentally as there is insufficient knowledge of the mechanical properties of soft tissues which would permit determination of the mechanical limits from anthropometric data or nondestructive tests. The most direct and reliable approach to establishing allowable mechanical limits would be to conduct tests on human subjects which are representative of the population to be protected. In these tests, the subjects would be exposed to forces which would simulate the loads applied during flailing. Limits could be established by varying the applied loads and noting the subsequent level of injury which results. This methodology is clearly unacceptable because it results in injuries to live subjects.

An alternate approach is to conduct mechanical tests on specimens obtained from human cadavers. This, however, requires extrapolation of the resulting data to the conditions of interest. The extrapolation must be performed in three major areas:

1) It is necessary to consider the differences in mechanical limits between the population of cadavers, upon which the tests are performed, and the human population to be protected.

2) It is necessary to extrapolate the test conditions used in the laboratory to the loading environment to which the population is subjected.

3) It is necessary to relate the mechanical failure criteria used in the laboratory to functional injury levels.

At present, inadequate experimental data exists to establish more than a few limited limb flail design criteria. For this reason our main purpose here is to discuss the major factors which must be considered in extrapolating laboratory data to the operational conditions for which the design limits are specified. This will be accomplished in the following order:

1) A discussion of the mechanical properties and load-elongation behavior of ligaments,

2) review of the mechanism of ligament failure and,

3) consideration of the factors which influence the mechanical properties and failure mechanisms. This includes the effects of specimen storage, test strain rate, test temperature, disuse factors, age, and variations with species.

Finally, torsional failure data obtained on human cadaver knee preparations will be presented and the results discussed in terms of its application to the establishment of limb flail design limits.

MECHANICAL PROPERTIES OF LIGAMENTS

INTRODUCTION

In order to fully understand joint mechanics and the mechanisms of joint injury, it is necessary to understand the mechanical properties of the ligaments under loading conditions which are typical of both normal function and trauma. Many studies have been performed to establish the mechanical properties and behavior of tendons (1-14), ligaments (15-36), and other collagenous structures (37-50). That these biological tissues are viscoelastic, exhibiting creep, stress relaxation and strain rate sensitivity is now well known (1,17,19,22,37-42,47,52). While these studies have provided important information, our knowledge is still incomplete. The unique anatomical characteristics of individual ligaments, such as fibril organization and insertion site characteristics, prevents generalization to other ligaments. In addition, there appears to be important relations between macroscopic mechanical behavior and tissue microarchitecture (22,26,41,42,50,53). That is, the mechanical behavior depends not only on the material properties of the collagen fibrils, but also upon the geometric arrangement of the fibrils and fiber bundles, the proportioning of the different types of fibrous constituents, and the relatively unknown effect of the ground substance. In the analysis of ligament properties, it is often difficult to separate the effect of these different factors. For this reason, a great deal of caution is required in generalizing the mechanical properties presented here to other ligaments.

STRUCTURE

Composition and Organization

Ligaments are dense connective tissue structures which join together adjacent bones at their ends, thereby helping to maintain skeletal alignment and body shape. The primary substance of ligaments is intercellular fibers which are aligned with the long axis of the ligament. The fibers are capable of resisting tensile forces, but offer little resistance to compressive, bending, and torsional loads. There are two primary types of connective tissue fibers, collagen and elastin. Collagen predominates in most ligaments and is the main tensile force transmitting structure in the human body. Elastin appears primarily in those special situations, such as the spinal ligaments, where the ability to transmit a tensile force must be combined with the ability to undergo large strains (greater than 50%). In addition to the intercellular fibers, ligaments contain a ground substance composed of proteoglycans and water which surrounds the fiber, a vascular bed, and a cell population. In contrast to other dense connective tissues, such as tendons and cartilage, the vasculature of many ligaments is well developed and profuse. While the cells that appear are varied, the predominant cell is the fibroblast which manufactures the fibrous proteins, collagen and elastin.

It is generally conceded that the most fundamental molecular structure in collagenous tissues is the tropocollagen molecule (6,54). The molecule is composed of a series of coded amino acid chains arranged into a right-handed superhelix. Five such parallel tropocollagen molecules are then staggered to form a microfibril (35-1,000 Å) (6,54,55,56).

Beyond this level, the classification system has been debated. Some have suggested that a lattice of these microfibrils form a subfibril while others have distinguished only the fibril, the finest strand of collagen visible under the light microscope. These fibrils are in turn embedded in an interfibrillar matrix (acid mucopolysaccharide). The fibril has been identified as the basic load-bearing unit in such connective tissues as ligaments and tendons (55,57).

A waveform appearance may be observed under the light microscope when viewing tendons and ligaments (Figure 1). The crimping has been observed in the collagen fibril and is thought to have evolved by a buckling phenomena of matrix impinging on the fibrils during formation (57,58). Recently it has been shown that the crimp is a planar zig-zag pattern (6,58). The crimp has been thought to influence the initial loading of collagen through an unfolding mechanism (6,57).

Figure 1. Photomicrograph of collagen fiber bundles from human semitendinosus tendon in longitudinal section (300 X). Note the crimped pattern of the fibers.

The next higher element in the structural system in the fiber, which is a bundle of parallel fibrils held together by matrix. The fibers may be straight or wavy or branch at acute angles (55). Though the length is unknown, the fiber is believed to approach the length of the whole tendon (48).

The fiber (primary) bundle is a collection of fibers enclosed in fibrous connective tissue known as endotenon. A group of these primary bundles is called the fascicle and is surrounded by another sheath, the epitenon (14,48). The fascicle, which still maintains a crimped appearance due to the axial alignment of fibrils, represents the smallest collagenous structure which may be mechanically tested.

The macroscopic tendon or ligament is a collection of fibers or fascicles enclosed in a paratenon sheath. The differences which occur in the response of various tendons and ligaments are believed to be the result of variations in the arrangement or alignment of fibers or fascicles within the tissue (14). Among connective tissues, tendons have been found to have the most parallel fiber arrangement. Ligaments, however, may possess either parallel or oblique fiber arrangements depending upon their role in restraining forces at a joint (48).

Insertion

The insertion of ligaments into bone commonly occurs through four well defined zones which have been described by Cooper and Misol (59). The zonal arrangement of the insertion basically represents a change in the composition of the medium surrounding the collagen fibers. This zonal arrangement for the insertion of a rhesus anterior cruciate ligament into the tibia is shown in Figure 2. Zone 1 corresponds to the ligament body where the medium surrounding the collagen fibers

Figure 2. Photomicrograph of a Rhesus anterior cruciate ligament-tibia insertion of one specimen that failed by cleavage through cancellous bone. The zones of the bone-ligament interface are described in the text. Zone 1 = ligament Zone 2 = fibrocartilage; Zone 3 = mineralized fibrocartilage; and Zone 4 = bone (H&E Stain, X9).

is primarily ground substance composed of glycosaminoglycans. In the second region Zone 2, the cells assume the appearance of chondrocytes found in cartilage being round and regularly spaced in rows or columns. Zone 3, mineralized fibrocartilage, is separated from Zone 2 by a well defined blue staining line. The last region; Zone 4, is composed of lamellar bone. The collagen fibers lose their parallel arrangement in this region as they interdigitate with the haversian systems.

TEST PREPARATIONS

Most of the ligament properties presented here were obtained from studies conducted using femur-anterior cruciate ligament-tibia preparations from either rhesus monkeys (22-26) or humans (26). Mechanical tests to failure were performed with the knee flexed to 45^{o} and the axis of the ligament aligned with the direction of the applied joint separation. The bone-ligament-bone preparation has the advantage, as compared to the isolated ligament, of reducing problems associated with gripping, such as slipping of the ligament within the grips, and the generation of stress concentrators which produce premature failure at the grip-ligament interface. An additional advantage of the bone-ligament-bone preparation is that the mechanical properties of all components of the unit are studied together in the same relationship they have in the body. Thus, the combined interaction determines the properties of the structure as *in vivo*. To a lesser extent the use of the bone-ligament-bone preparation also improves the uniformity of loading over the ligament cross-section. This advantage results from the fact that all of the ligament fibers are attached to the bone. A mechanical grip, on the other hand, cannot transfer load directly to the fibers in the center of the ligament, but must rely upon shear between the fibers to transmit the load. Use of the bone-ligament-bone preparation does not automatically insure uniform loading. Ligament microgeometry combined with improper orientation of the bones can result in preferential loading to one portion of the ligament.

In some ways, a bone-ligament-bone preparation is more complicated and requires greater care in testing than the isolated ligament. Failure can occur via a ligamentous tear, an avulsion fracture of the bone under the ligament insertion, or cleavage between the ligament and bone at the insertion site (22). Commonly, failure will occur by a combination of these modes. It is therefore necessary to consider not only those factors which affect the ligament, but also those which affect the insertion or the underlying bone. Some of the important factors which will be discussed here are the effects of storage by freezing, rate of deformation (strain rate) (22), antemortem disuse and immobility states (23,24), and age (26).

LOAD-ELONGATION BEHAVIOR

A typical force vs. elongation curve for a cruciate ligament preparation is shown in Figure 3. These curves commonly exhibit a concave toe region at the onset of elongation so that the preparation becomes stiffer as it is stretched. This behavior is thought to be due primarily to the straigtening of the crimp in the collagen fibrils discussed earlier. Other factors such as an increase in the number of taut load bearing fibers and improved alignment of fibers with the

loading axis also contributes to an increased stiffening with increased strain.

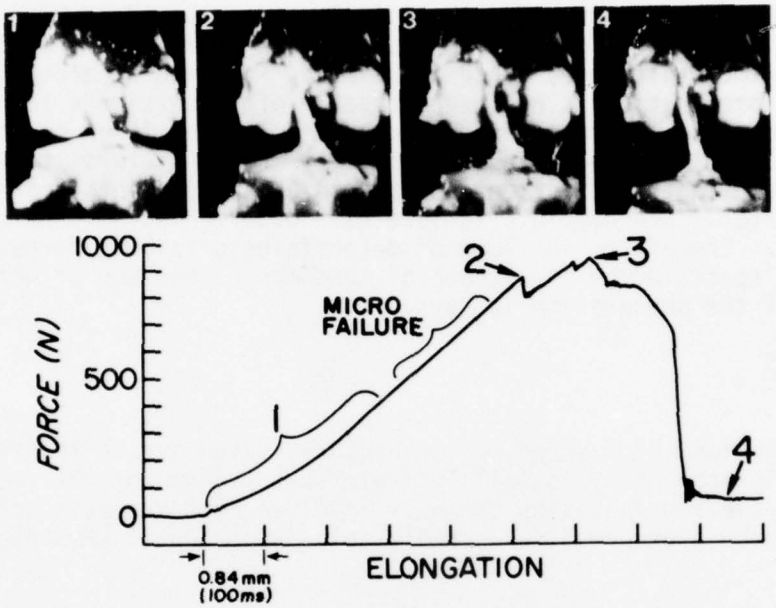

Figure 3. Oscillograph record of force versus time for a tension test to failure of a Rhesus femur-anterior cruciate-tibia preparation. A constant distraction rate was used so that the time axis was proportional to specimen elongation. The photographs, obtained from high speed movies taken during the test, show the preparation at four stages in the test.

Following the non-linear toe there is a nearly linear region up to the first significant failure which is identified by the sudden drop which occurs in the force. In this linear region the collagen fibers do not have the crimp seen in the toe region, the crimp having been completely straightened by the elongation. The end of the linear region is referred to as the linear load point and it signifies the approximate start of major failure. The use of a yield force is avoided due to the fibrous nature of the ligament and the difficulty in determining the onset of permanent elongation in viscoelastic materials. The drop in force at the linear load point may be due either to rupture of collagen fibers in the body of the ligament, failure at the ligament-bone interface, or to partial avulsion of the underlying bone. In general it is impossible to determine from the load-elongation curve the origin of the failure. In some specimens the onset of major

failure is not indicated by a sudden drop in force, but by a reduction in stiffness as with a yielding metal. In this case a linear load point can be determined by the slope offset technique used to determine offset yield stress in common engineering materials. The amount of the offset used for biological specimens is usually larger than that employed for metals, ranging from 0.2 to 2.0% elongation.

Past the linear load point, additional failures can be identified by the subsequent drops in the force trace. This successive failure of the ligament continues until a maximum (ultimate) force is reached, followed by a continued serial failure until the preparation is no longer capable of supporting a load. In general, a failure force, corresponding to complete loss of fiber continuity within the ligament, cannot be identified. High speed motion pictures taken during the test demonstrate that the ligament appears to be grossly intact and continuous after the maximum force and complete failure have occurred as will be discussed in more detail later. Therefore, in place of determining a failure force, complete failure should be specified by the amount of specimen elongation at which the load bearing ability of the preparation is lost.

FAILURE MECHANISMS

Visual examination, high speed motion pictures taken during testing, and microscopic histological analysis, all indicate that failure of the femur-anterior cruciate-tibia preparation commonly involves both the osseous and ligamentous components in a progressive unpredictable manner until complete failure occurs.

Microscopic examination of failed preparations using standard histological techniques demonstrate three primary failure modes:

1. Ligamentous failure, best described as failure by a pulling-apart of collagen fibers (Figure 4). Rupture of collagen fiber bundles occurs throughout the body of the ligament and is not localized to one specific area. The geometric peculiarities of the microarchitecture determine which fiber bundles are under the greatest deformation and stress during loading. The serial rupture of ligament fibers seen from the load-elongation curves and from high-speed motion pictures may account for the microscopic observation of ruptured fibers being adjacent to intact fibers. The pulling-apart of ruptured fibers suggests that little in the way of cohesive properties exists between the major fiber bundles. A more explicit definition of the failure mechanism of collagen fibers must await the result of detailed studies on the ultra-structural changes which occur in fibrils and microfibrils under various loading conditions.

2. Bone avulsion fracture, the second failure mode, occurs most commonly through the cancellous bone immediately beneath the more dense cortical bone at the site of the ligament insertion (Figure 2). Minute gritty material, sometimes just barely palpable on the end of the ligament after failure, usually proves to be fragments of bone, the failure having occurred through the cortical bone deep to the fibrocartilaginous zone of ligament insertion.

3. Cleavage at the ligament-bone interface is the third and last common failure mode. Failure is observed throughout the four zones of ligament insertion

although there is a definite prediliction for the cleavage line to occur through

Figure 4. Photomicrograph of a Rhesus anterior cruciate ligament that failed by rupture of collagen fiber bundles. Notice failure occurs at different points along the ligament giving the gross appearance of a "mop end" (Trichrome, X45).

the zone of mineralized fibro-cartilage (Zone 3) at or just distal to the blue staining line (Figure 5). Failure at the ligament-bone interface in one series of tests, was observed microscopically in six of twenty-two specimens (22). This mode represented only a minor part of the failure process, the major failure having occurred by either one or both of the other modes (ligamentous or bone avulsion fracture). Failure through the fibrocartilaginous zone was therefore never the major mode of specimen failure, but not infrequently accompanied the other two modes of failure. The gross appearance of specimen failure at the bone-ligament junction usually proves upon histological examination, not to involve the zonal interface, but rather to occur some distance from the interface either in the body of the ligament or deep in the cortical bone.

Although it is usually possible to classify the major mechanism of specimen failure histologically, it is the rule to observe other minor modes of failure

involving, to different degrees, all portions of the bone-ligament-bone unit.

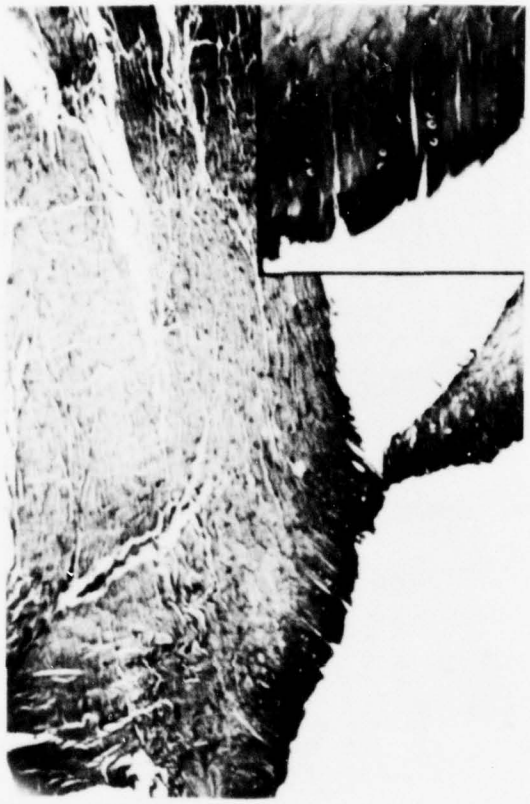

Figure 5. Photomicrograph of a Rhesus anterior cruciate ligament that failed through Zone 3, mineralized fibrocartilage of the ligament bone interface. Insertion shows columnar arrangement of chondrocytes (periodic acid-Schiff, X80).

Thus it is not unusual to find microscopic evidence of minor to moderate ligament disruption associated with major bone avulsion failures, or vice versa.

On empirical grounds, the zonal arrangement at the site of ligament insertion should be advantageous in producing a gradual change in mechanical properties thereby decreasing the expected stress-concentration effect of the ligament's insertion into the stiffer bone structure. This should also protect against fatigue failure and against shear failure at the bone-ligament junction under in vivo loading conditions. The ligament at the insertion site may behave more as a true composite material than the remainder of the ligament because of the relationship between the ground substance and collagen network. The cartilaginous ground substance probably provides greater cohesion between fiber bundles and hence provides a mechanism for diffusion of load over the entire insertion site, thereby avoiding the deleterious effects of stress concentration. The effectiveness of the zonal

insertion in reducing local stresses can only be speculated on, since no information is available on how specimens would fail in its absence. Based on the microscopic analysis of specimen failure and the less frequent occurrence of failure through the fibrocartilaginous zone, the zonal insertion arrangement does appear mechanically advantageous.

Failure mechanisms of bone-ligament-bone preparations other than the anterior cruciate ligament unit exhibit both similarities and differences. Figure 6 shows typical load-elongation curves for the rhesus medial collateral ligament and two

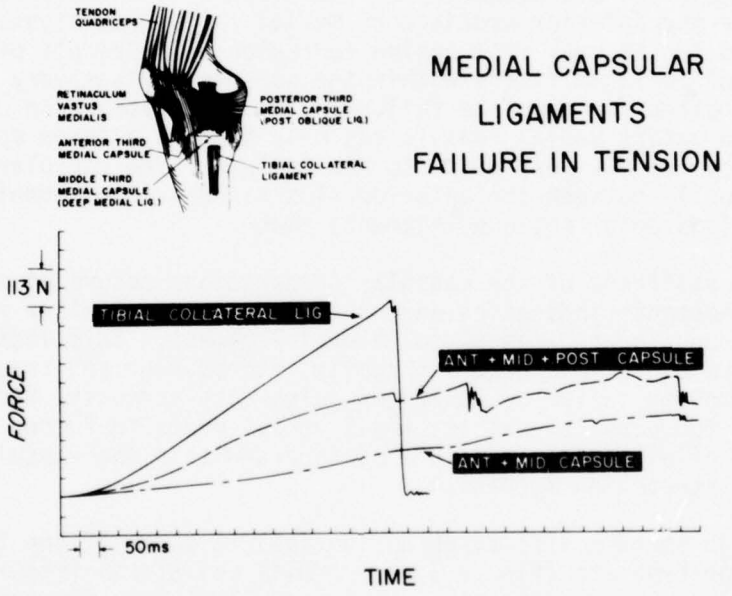

Figure 6. Typical oscillograph record of force vs. time for failure tests on the medial collateral ligament and two capsular preparations. A constant distraction rate was used so the time axis is proportional to specimen elongation. The MCL supports the largest forces and fails abruptly. This is in contrast to the progressive failure of the ACL shown in Figure 3 and the two capsular preparations. Note the reduction in stiffness which occurs for both the MCL and capsule prior to significant failure indicated by the sudden drop in resisting force. This is indicative of a progressive microfailure process which occurs prior to macroscopic failure.

rhesus medial capsular preparations which were failed in tension with the knee flexed to 45 degrees. The two capsular preparations are the entire medial capsule back to the mid-popliteal region and just the anterior plus mid medial (deep MCL) capsule. In both capsular preparations, the medial collateral ligament is cut.

The medial collateral ligament develops the largest force when elongated and fails abruptly. This is in contrast to the progressive serial failure of the femur-ACL tibia preparation shown in Figure 3. The orientation of the collagen fiber bundles within the MCL, as compared to the ACL, appear more uniform both visually and under low power magnification. The relative positions of the bones in the test fixtures, along with collagen fiber orientation and the relative length of the individual fibers determine the sequence in which the fibers are loaded and fail. The abrupt failure of the medial collateral ligament is believed to be a result of both the uniform orientation and uniform loading of individual collagen fibers and fiber bundles.

In contrast, the fiber microgeometry of the capsule is considerably less organized than either the anterior cruciate or medial collateral ligament. Both fiber orientation and length vary from region to region. One result of the more random organization of collagen fibers within the capsule is that very large joint displacements are required for complete failure. The load-elongation curve shown in Figure 6 shows the entire medial capsule can only support a force approximately one-half that borne by the MCL just prior to MCL failure. The capsular strength is divided nearly equally between the anterior plus mid one-third combined and posterior one-third (posterior oblique ligament) alone.

A reduction in stiffness of the capsular preparations occurs at small to moderate joint displacements indicating an onset of failure. Similar reductions in stiffness are also found for the medial collateral ligament. This indicates that while complete failure of the MCL occurs abruptly, microdamage and trauma occurs serially prior to complete failure. At larger joint displacements, the load vs. elongation curve for the capsule exhibits small serial drops in force. This indicates the occurrence of significant failure of fibers within the capsule by a progressive tearing the stretching mechanism.

Stills from high speed movies taken during failure tests of the two ligament preparations, anterior cruciate (Figure 3) and medial collateral (Figure 7) show that loss of continuity occurs well after complete failure when the preparation is not able to support a load. The large joint displacements required for loss of continuity combined with the irregular mop end appearance of the failed ligament indicates that a shear pull-apart failure occurs between fibers after fiber rupture. The presence of gross continuity and the lack of visual evidence of failure even after the force-elongation curve, is also found for capsular preparations.

MICRO-TRAUMA

As a part of the study of failure mechanisms, anterior cruciate ligament preparations were subjected to a force approximately one-half the typical failure force without any observable failure or change in force-elongation behavior indicating failure. This test simulates functional conditions in which a ligament or collagenous structure is subjected to a significant load or elongation that is still

Figure 7. Composite of three stills made from high speed movies taken during a failure test of the medial collateral ligament. On the left at the onset of loading, in the center after complete failure and loss of load-bearing capacity. Loss of continuity required joint displacements larger than shown on the right.

well within the failure limits. The specimens were immediately unloaded and examined by scanning electron microscopy. Two ligament-bone units were loaded in this manner and two matched preparations from the opposite extremity served as controls. Scanning electron microscopy of the loaded specimens showed complete failure had occurred in some collagen fiber bundles as seen in Figure 8a. Also shown is a second mechanism of microfailure, Figure 8b, where separation of fibers within a fiber bundle has occurred with disorganization of interconnecting fibers. In addition, the fibers within the larger bundle have a straightened appearance indicating that significant elongation past their elastic limit has occurred.

Figure 8a. Scanning electron microphotograph of an anterior cruciate ligament after it was loaded to one-half its anticipated maximum load without evidence of failure. Microfailure occurrs. A ruptured collagen fiber is shown in this picture.

Figure 8b. Scanning electron microphotograph of an anterior cruciate ligament after it was loaded to one-half its anticipated maximum load without evidence of failure. Microfailure occurs. Separation between fiber bundles and fibers is shown in this picture.

In Figure 9, a large collagen fiber bundle is shown at the site where failure occurred. The jagged appearance results from failure of small collagen fibers

Figure 9. Scanning electron photomicrograph of a collagen fiber bundle at the plane where failure occurred. The failure of collagen fibers at multiple levels within the fiber bundle demonstrates the pull-apart failure process. The arrows indicate a rich supply of vessels lying on the surface of the fiber bundle with perpendicular branches supplying the interior of the fiber bundle. Failure of the vessels has occurred, however; just proximal to this plane, they appear relatively intact with little disorganization.

and fibrils at different levels within the fiber bundle. This indicates the existance of a shear pull-apart type of failure after initial rupture of the finer collagen fibers and fibrils within the fiber bundle. This is similar to the

rupture of fiber bundles at different levels throughout the ligament followed by a shear failure between the adjacent disrupted fibers. Of interest is the presence of blood vessels and smaller vessel tributaries at the failure site. The vessels can be seen traversing the failure site and have a relatively normal appearance almost up to the point where failure occurred.

FACTORS AFFECTING MECHANICAL PROPERTIES

EFFECTS OF FREEZING

One of the major problems which must be faced when testing biological tissues obtained from humans, is that it is not always possible to arrange for immediate testing when the preparation becomes available. For this reason it is often necessary to employ some form of storage until the test, usually refrigeration or freezing. The use of formaldehyde fixation which inhibits tissue lysis degradation, also alters the mechanical properties of collagen tissue and will not be considered further.

A review of the literature indicates that only few studies have been conducted to specifically examine the effects of storage mode (21,32,43,64-66). The data available indicate that there is no statistically significant difference between the mechanical properties of freshly tested preparations and those which have been frozen, stored for a period of time, and thawed prior to testing. While statistical differences are usually not detectable, the data do indicate that there is a tendency towards altered mechanical properties and reduced strength with frozen specimens.

To examine this question more closely, a study was conducted of the effects of freezing upon the mechanical properties of bone-ligament-bone preparations. Right-left pair preparations from 10 rhesus monkeys were failed in tension at a strain rate of 66%/sec. One-half of each pair was immediately frozen at -15^{o}C and stored for three weeks prior to testing. The other preparations were tested within two hours after sacrifice of the animal. The results of this study are shown in Table 1. These results are consistent with the previously reported data in the literature. While no statistical difference exists between those specimens which were tested immediately and those which were frozen prior to testing, there is a tendency towards a reduced maximum stress and energy to failure in the frozen specimens. It is likely that if all of the available data on the effects of freezing were pooled, a statistically significant effect of freezing would be obtained. The available data to indicate, however, that the magnitude of the effects of freezing is generally less than the normally occurring biological variability.

TABLE 1

EFFECTS OF FREEZING ON RHESUS FEMUR-ACL-TIBIA UNITS

PARAMETER	NOT FROZEN (N=10)	FROZEN (N=10)	PERCENT DIFFERENCE
Weight (Kg)	6.94 ± 0.574*	6.94 ± 0.574	0
Linear Load (newtons)	754 ± 112	679 ± 92.9	-10.5
Maximum Load (newtons)	859 ± 116	785 ± 95.8	- 9.0
Stiffness (K newtons/meter)	194 ± 29.8	190 ± 28.0	- 2.1
Energy to Failure (newton-meters)	3.20 ± 0.533	2.67 ± 0.575	-18.1
Linear Stress (MPa)	58.2 ± 8.3	55.0 ± 5.3	- 5.7
Maximum Stress (MPa)	66.1 ± 6.9	63.9 ± 9.0	- 3.4
Modulus (MPa)	184 ± 23.7	190 ± 30.6	+ 3.2
Energy Density (N-m/cc)	20.1 ± 3.4	17.7 ± 4.4	-12.7
Initial Strain (%)	8.57 ± 3.85	9.89 ± 1.60	+14.3
Strain to Linear Load (%)	39.6 ± 7.76	37.9 ± 4.00	- 4.4
Strain to Maximum Load (%)	47.9 ± 4.75	45.0 ± 6.14	- 6.2
Strain to Failure (%)	60.9 ± 7.83	61.1 ± 4.78	+ 0.3

*Values given are means ± standard deviation

STRAIN RATE EFFECTS

It is well recognized that ligaments, like other passive soft biological tissues, exhibit viscoelastic behavior, including creep, stress relaxation, and strain rate dependency (1,17,19,22,37-43,47,52). In vivo, the strain rate to which the ligaments are subjected varies greatly depending upon the ligament considered, the level of activity, and specific form of activity being performed. The wide variation in the in vivo dynamic loads seen by ligaments raises the question as to the functional significance of viscoelastic behavior and its effects on traumatic events. Most of the studies on the mechanical properties of ligaments and tendons have been conducted at strain rates which are very low and generally not representative of in vivo loading or trauma conditions (15-19,27,31,33-35). Even those few studies which considered the effects of strain rate have not conducted tests

which would simulate the in vivo loading rates expected in light or moderate activity.

In order to determine the effects of strain rate on the load-elongation behavior and failure mechanics of femur-anterior cruciate-tibia preparations Noyes et al. (22) tested 17 right-left pairs from male rhesus monkeys. One side was failed at a slow rate of 0.08467 mm/sec and the opposite side failed at a fast rate of 8.467 mm/sec. This corresponds to approximate strain rates of .65% sec and 65%/sec respectively. The faster rate produces a 10% elongation in slightly over 150 milliseconds. This is similar to the loading rate anticipated during moderate activity.

The results of this study indicate that the ligamentous portion of the bone-ligament-bone preparation exhibits only a small strain rate effect. The bony portion underneath the ligament insertion site, however, is sensitive to strain rate. This was observed from changes in the failure mechanics of the preparation.

This may be better understood by careful examination of Table 2 which presents the average load-elongation properties for the fast and slow groups, indepen-

TABLE 2

EFFECT OF STRAIN RATE ON SEVENTEEN MATCHED KNEE PAIRS

PARAMETER	FAST	SLOW	PERCENT DIFFERENCE
Linear load (newtons)	893 ± 204	779 ± 163	13.6[a]
Maximum load (newtons)	998 ± 165	806 ± 176	21.3[b]
Strain to linear load (%)	43.5 ± 12.2	42.7 ± 7.7	1.8
Strain to maximum load (%)	51.5 ± 6.7	43.7 ± 7.0	15.6[c]
Strain to failure (%)	57.1 ± 10.4	51.9 ± 10.1	9.5
Energy to failure (joules)	4.12 ± 1.09	3.01 ± 0.85	31.1
Slope at 10 per cent strain (KN/m)	157 ± 31	152 ± 31	3.3
Slope at 20 per cent strain (KN/m)	179 ± 44	168 ± 40	6.3
Slope at 40 per cent strain (KN/m)	142 ± 46	128 ± 30	10.4

[a] = $p < 0.05$

[b] = $p < 0.01$

[c] = $p < 0.005$

Absence of asterisk indicates to significant difference

dent of the failure mode, and Table 3 which presents the ultimate strength properties of both strain rate and failure mode (i.e., ligamentous vs. tibial

TABLE 3

EFFECT OF STRAIN RATE AND FAILURE MODE UPON MECHANICAL PROPERTIES

Strain Rate	Number	Failure Mode	Maximum Load (newtons)	Strain to Maximum Load (per cent)	Energy (joules)	Strain to Failure (per cent)
Fast	21	Ligamentous	964 ± 172	51.4 ± 7.5	3.91 ± 0.87	61.0 ± 8.9
	9	Tibial avulsion fractures	982 ± 189[b]	51.6 ± 7.0[b]	3.77 ± 1.29	55.0 ± 12.4
Slow	8	Ligamentous	913 ± 104	47.8 ± 6.0	3.92 ± 0.95[a]	59.0 ± 9.3[a]
	16	Tibial avulsion fractures	826 ± 197[b]	45.6 ± 7.4[b]	3.03 ± 0.91[a]	50.9 ± 8.0[a]

[a]Mean value significantly different from that of other failure mode at the same strain rate.

[b]Mean value significantly different from that of the same failure mode at the other strain rate.

Table 2 shows that the only parameters in which a statistically significant difference occurred between the fast and slow groups are those parameters which relate to the failure strength of the preparation, such as linear load, maximum load, strain to maximum load and the energy to failure. Conversely, prefailure properties like stiffness were not greatly affected by a one-hundred fold change in strain rate.

The response of the ligamentous portion of the preparation predominates in the prefailure region while the response of the bony portion predominates in the strain-rate sensitive failure region as seen in Table 3. Additional preparations were tested and included in this Table to increase the number of preparations in each failure mode, permitting better statistical comparisons. When the 21 preparations which failed by ligamentous tear at the fast strain rate are compared to the eight preparations which failed by ligamentous tears at the slow strain rate,

no statistical difference is found in any of the parameters. Clearly, when the response of the bone-ligament-bone preparations is completely dominated by the ligament alone, a one-hundred fold change in the strain rate does not significantly affect either the load-elongation properties or failure strength. It may be concluded from these results that the strain rate dependency of the ligament, over the range tested, is less than the normal biological variability.

The bony portion of the preparations, on the other hand, is significantly affected by strain rate as can be seen from Table 3 and Figure 6. In those preparations which failed by tibial avulsion fraction, there was a significantly smaller maximum load and smaller strain to maximum load at the slow rate vs. the fast rate. Note that for the fast rate there is no statistical difference between the preparations which failed by tibial avulsion fracture and those that failed by a ligamentous tear. At the slow rate, however, preparations that failed by a tibial avulsion fracture failed at a smaller strain and lower energy than preparations which failed by a ligamentous tear.

The major failure modes of the 32 specimens tested at the fast deformation rate and of 29 specimens tested at the slow deformation rate are shown in Figure 10. At the fast rate, two-third of the specimens showed a ligamentous type of failure whereas 28% failed by tibial avulsion fracture. At the slow rate, the reverse was true; the most common type of failure was tibial avulsion fracture, occurring in 57% of the specimens.

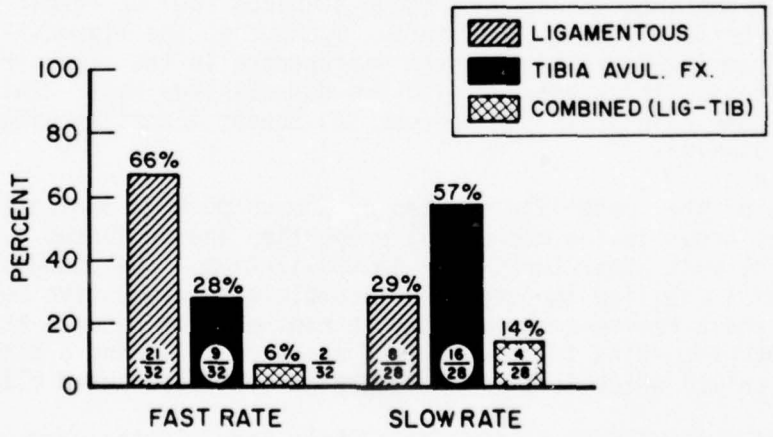

Figure 10. The major failure mechanism for thirty-two bone-ACL-bone units tested at 65%/second and twenty-eight units tested at 0.65%/second. The difference in failure mechanism between fast and slow tests is statistically significant ($p < 0.05$) by the chi square test ($\chi^2 = 8.22$).

Ligamentous disruption occurred in 29%. The difference in failure mode between the fast and slow rate groups was statistically significant at the $p < 0.05$ level.

DISUSE

The deleterious effects of immobility and disuse states on bone, joint, and soft tissue structures have been demonstrated in a number of studies involving experimental animals and man (2,20,23,68-90). The cause of the disuse effects, while not fully understood, are thought to be related to many factors; including the nature of superimposed disease process, the form of immobilization as well as its duration and severity, and not least of all, the overall activity state of the animal or man.

Little information has been published concerning the effect of immobility on the functional properties of ligaments or the permanency of such alterations once they occur.

To study the effects of immobility upon the mechanical properties of ligaments Noyes et al. (23,24) tested anterior cruciate bone-ligament-bone units from recently captured wild primates. Captivity period was kept to the shortest possible time to minimize the influence of disuse effects due to cage confinement. Under these experimental conditions, the failure properties of the ligament itself may be analyzed during the mechanical test for it participates in the failure process. This is often not possible in ligament units of confined laboratory animals in which failure at the bone attachment site occurs well before the ligament has been subjected to failure loads. For example, inactivity in dogs as a result of the cage confinement has been shown by Laros (20) to result in premature failure of the superficial tibial collateral ligament at the ligament-bone attachment. Barfred (2) found significant differences in the strength and failure mode of achilles tendon units between wild and domesticated rats. This was presumably due to disuse changes in the bone at the tendon insertion site in the confined domesticated animals.

The results of the immobility studies on Rhesus monkeys indicate that significant alterations occur in the mechanical properties and projected functional capacity of a ligament unit after short-term immobilization. The changes are related to the severity of immobility imposed with incomplete recovery five months after resumed activity and a return to normal twelve months after resumed activity. The change in ligament properties to be described occurred following a disuse state in the experimental animal which is similar to that often encountered clinically.

Two forms of immobilization were used; whole body plaster casts and cylinder cast immobilization of one lower extremity (23,24). In one large series, 69 male rhesus were divided into four groups. The control consisted of 28 animals. The immobilized groups consisted of fifteen animals placed in whole body plaster casts for eight weeks. An additional twenty-one animals were first immobilized for eight weeks and then returned to activity for reconditioning. Eleven animals were reconditioned for a total period of five months while the remaining ten animals were reconditioned for twelve months. Mean weight change after immobility was -12.3% for the immobilized group, +1.5% for the five-month and -1.4% for the

twelve-month reconditioned group. After reconditioning, the animals were 103% and 144% of their initial weight for the five and twelve-month groups, respectively.

Nearly identical initial mean weights for the control and experimental groups were obtained by selection and matching of the animals assigned to each group. This selection minimized the necessity of normalizing the strength parameters by animal weight as has often been done previously. Homogeneity of the animals in the control and experimental groups was demonstrated by correlation coefficient matrix analysis of the variables, captivity time and animal weight, against the mechanical strength parameters of the ligament unit.

In a second series, the right lower extremity of five rhesus monkeys was immobilized for eight weeks with a cylinder cast that extended from the thigh to just above the ankle. Variations in the ligament size were accounted for by direct measurement of length and cross-sectional area. The effects of the immobilization upon ligament material properties as determined by paired tests between left and right limbs was consistent with the changes measured in force elongation curve where the variation in size was not accounted for.

The effects of immobility on the average force-elongation curve of those animals subjected to whole body immobilization are shown in Figure 11. Only speci-

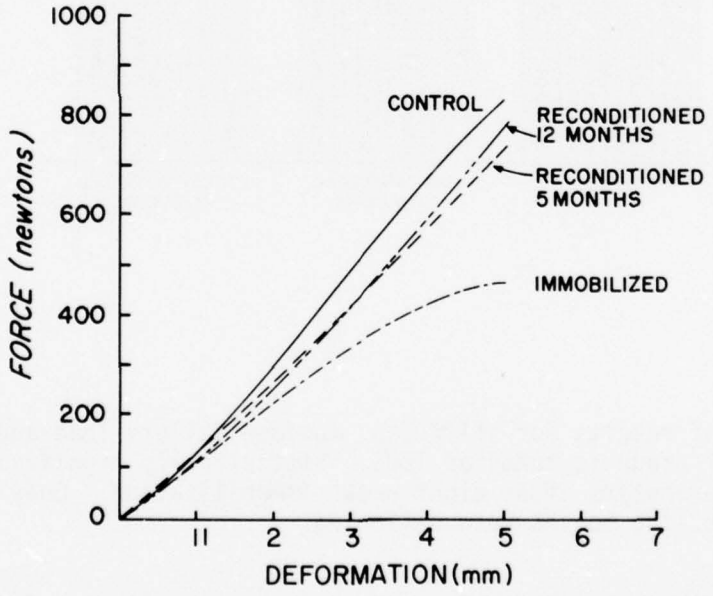

Figure 11. Summary force-deformation curves for femur-ACL-tibia from control, immobilized and reconditioned rhesus monkeys. A significant decrease in stiffness occurs after eight weeks immobilization and a significant recovery occurred after five months reconditioning. Curves obtained by least squares cubic fit of data to five millimeters elongation.

men response in the prefailure region is shown because failure mechanism varied between experimental groups as will be discussed. Regression analysis was used to obtain least squares cubic fit to each test and for all the tests within a single group. Statistical analysis of the summary curves indicated a significant decrease in stiffness to 69% of control after eight weeks immobilization and a return to 93% of control after five months reconditioning.

The changes in ultimate strength (maximum load), energy failure and stiffness are summarized in Figure 12. The maximum load and energy, like stiffness, show

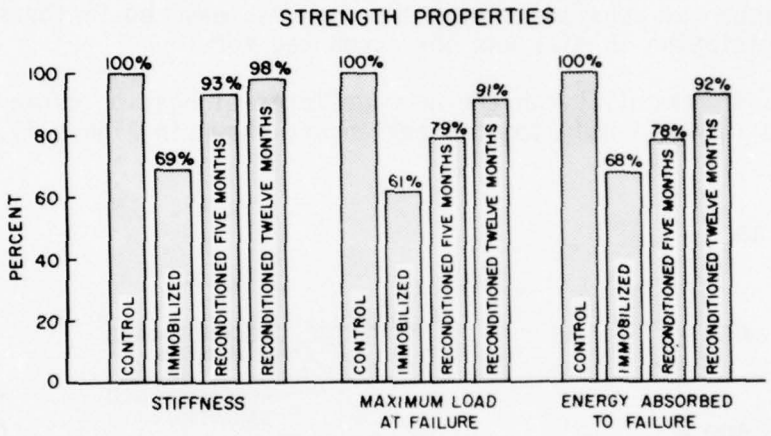

Figure 12. Summary of results for stiffness, maximum failure load and energy to failure. The control group is taken as 100%. Statistically significant decreases occur in all three parameters after eight weeks immobilization. Only partial recovery occurred after five months reconditioning.

statistically significant decreases after eight weeks immobility. For the five-month reconditioned group, the maximum load and energy only partially recovered. After one year of reconditioning, no statistical difference existed in maximum load and energy between the control and re-conditioned specimens.

To permit comparison of failure modes between the animal groups, each failure was classified into the major or predominant mechanism of failures as shown in Figure 13. The combined mechanism refers to those cases in which both ligamentous

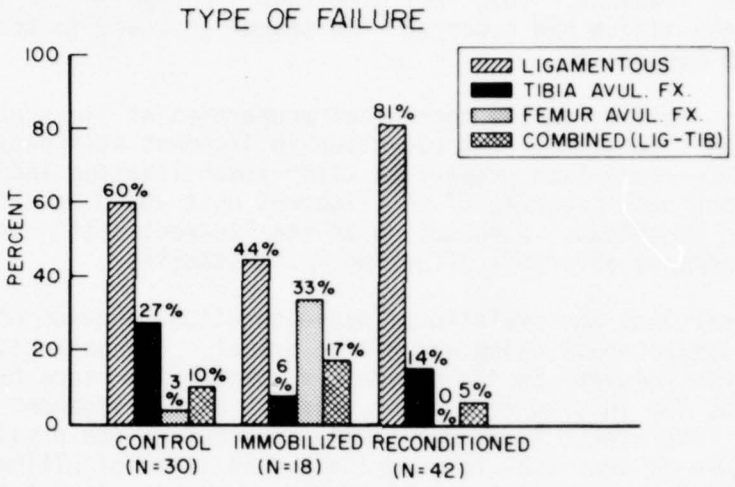

Figure 13. Major failure mechanism of femur-ACL-tibia units in the control, immobilized and reconditioned groups (five and twelve month combined). The most common failure mechanism is ligamentous in each group. An increase in femoral avulsion fractures occurred in the immobilized groups but not in the reconditioned group.

failure and tibial avulsion fracture occurred. The most common type of failure in all groups was by a ligamentous tear. Of significance, however, is the increase in frequency of femoral avulsion fractures in the immobilized group, where six out of eighteen specimens failed by this mechanism. Three other specimens had minor avulsion fractures at this site. This change in the pattern of failure between the control and immobilized group was statistically significant ($p < 0.05$). The five month reconditioned group showed a return (no statistical difference) to the control distribution of failures due to the low incidence of osseous failures in these preparations.

Effect of Immobility on the Ligament Itself

Failure by a major ligamentous mode occurred in 44% of the specimens following immobilization by total body plaster casts. A significant decrease in failure strength of these ligament units as compared to controls indicated that the ligament

itself had been affected. Additionally, failure of the ligament was observed in the high-speed motion pictures both prior to and in conjunction with the major osseous types of failure. In animals that had one limb immobilized by a cylinder cast, a statistically significant decrease of 22% was found in both the elastic modulus in the linear prefailure region of the force-elongation curve, and in the ultimate stress of the ligament. This indicates that a change in the material properties of the collagen tissue had occurred. No change occurred in the cross-sectional area of the ligaments.

The effect of immobility on the mechanical properties of ligament has not been well recognized in the literature. The reduction in ligament stiffness and simultaneous change in force-elongation properties after immobilization indicate that the projected in vivo functional capacity of the ligament unit would be altered. Under equal in vivo loading conditions, a reduction in the ligament stiffness will allow greater joint displacements adversely affecting joint stability.

It is of interest that the prefailure load-elongation behavior of the ligaments after five months reconditioning was nearly normal. In contrast, there had been only an incomplete recovery in the ultimate strength parameters by that time. This means that the in vivo functional capacity of the ligament unit would be near normal after five months resumed activity, as long as the physiological loading conditions were at less than failure loads. In terms of ultimate strength properties (maximum load, energy absorption) of the ligament unit, it required twelve months of resumed activity for recovery to control values.

Effect of Immobility on the Osseous Component

The decrease in ultimate strength of the ligament unit after immobilization, in those cases that failed by avulsion fracture, may be explained by resorption of Haversian bone and resultant weakening of the cortex beneath the ligament insertion site. This fact underscores the need to appreciate the potential for such effects in those conditions associated with reduced bone mass. Osteopenia can often be observed in roentgenograms of the lower extremity following injury and plaster immobilization as well as in a variety of other clinical disorders. It is not commonly realized that such bone loss may affect the strength of the cortical bone beneath the ligament attachment site and therefore, the strength of the ligament-bone unit as a whole. It is well known that loss of bone occurs starting in the fourth decade of life. It would be expected that the loss of bone might produce an age-related change in the failure properties of ligament-bone units.

As will be discussed in more detail later, studies of human anterior cruciate ligament-bone preparations show that older cadaveric and above-knee amputation specimens most commonly fail by bone avulsion fracture. In contrast, ligament preparations obtained from young adult trauma victims showed a greater ultimate strength with failure occurring throughout the ligament. These factors must be taken into account when experimental data obtained using cadaveric and amputation specimens are extrapolated to in vivo failure conditions.

After five months reconditioning, there is an absence of femoral avulsion fractures. This contrasts with the frequent occurrence of this type of failure in

specimens from the immobilized group. The change in frequency of osseous failures suggests a return of strength to the underlying bone at the attachment site of the ligament. This correlates histologically with the finding of active Haversian and periosteal bone formation and the filling-in of the prior resorptive spaces. In a parallel study using this same group of animals, measurements of tibial and femoral long-bone strengths showed a deleterious response to immobility similar in magnitude to that of the ligament unit. Complete return to normal bone strength (maximum failure load) occurred by five months. The results as a whole, show the different effects of immobility upon the mechanical behavior of the individual components of a functional bone-ligament-bone unit. When defining the mechanical integrity of ligament-bone units, it is important to consider all components and not simply, as is traditionally done, only the ligament itself.

Immobility and Joint Contracture

It is well appreciated that immobility can lead to joint contracture. The process by which this occurs is poorly understood. Qualitative as well as quantitative changes in the periarticular collagenous structures have been described (14,36,47,84,86,93-95). The deposition of collagen at certain points in relation to the capsule and ligament structures may restrict joint motion by not being properly aligned and may produce an effective shortening of these structures. A number of other mechanisms may be operative which have been recently summarized (70, 72,89).

It is important to separate the mechanical changes in the ligament unit described here, from the other concomitant effects of immobility which more specifically relate to the problem of joint contracture. In many knee specimens, the presence of a flexion contracture of the knee following immobilization was noted. This appeared to be due to an apparent shortening and contracture of the posterior capsular structures. The restriction to knee extension (that is, the relation between the torque applied to the angle of joint displacement) represents in this context an increase in joint stiffness (89). This emphasizes the difference between the posterior capsular ligaments and the anterior curciate ligament in terms of their response to immobility and the different clinical implications of such changes in terms of joint function. Further studies are required to define the effects of immobility on collagenous tissues, both in terms of structural properties as well as the material properties of the collagen fibers themselves.

AGE RELATED CHANGES

While mechanical studies of ligaments from animal models have provided much valuable information, extrapolation of the data to man is difficult due to differences which exist in anatomy, gait patterns, body metabolism and biochemistry. These difficulties have led to mechanical studies on human ligamentous tissue (19,21,26,33). Strength data on human ligaments is required, not only for the development of protective devices for high mechanical stress environments, but also for the design of artificial ligaments and total joint replacements.

Studies employing human tissues are more difficult to conduct than those using experimental animal tissue because of the problems involved in specimen acquisition. Prior knowledge of human specimen availability is rare and it is usually not possible to conduct the tests immediately after removal. Human specimens are usually stored frozen until just prior to testing. A second difficulty

associated with acquisition is the use of specimens obtained from older cadavers and from amputations such as in cases of chronic vascular insufficiency. Ante-mortem activity and disuse states cannot be controlled in human specimens and post-mortem degeneration may occur between the time of death and the removal of the specimen.

One important factor which can affect the mechanical properties of ligaments is age. Many of the data reports of human ligament strength appear low in comparison to the strength of rhesus ligaments (19,33). It was also noted that failure often occurred by bone avulsion fracture. This type of failure may predominate in animal preparations if there is a disuse atrophy or if artificially slow strain rates are employed in testing (22). In both cases, the ligament does not fully participate in the failure process. The resulting data is therefore not an indication of the true strength of the ligament.

To study the effects of age, 28 human femur-anterior cruciate-tibia preparations were tested in tension to failure (26). The methodology employed was identical to test methods used in prior studies, employing rhesus ligament-bone units with two exceptions. First, a slightly faster strain rate (100% per second vs. 66% per second) was employed and second, the specimens were frozen until just prior to testing. The higher strain rate was used to better simulate in vivo loading and because of the availability of a higher strain rate testing system. To study the effects of freezing, left-right pairs of rhesus ligament-bone units were tested, one immediately after animal sacrifice and one after three weeks of storage at -15^{o}C. As discussed earlier, no statistically significant difference was found in the strength of fresh vs. frozen specimens.

Of the 28 human specimens, eight were obtained from trauma victims, thirteen were obtained from amputations for vascular insufficiency and seven were obtained from donors with known disease states. The specimens from the trauma victims were considered to be free from ante-mortem disease factors which could affect the measured mechanical properties. The other specimens were considered potentially affected by disuse, disease, or ischema.

Figure 14 shows the force-time curves for two specimens obtained from trauma victims one age 22 and one age 50. Since a constant joint displacement rate was used in testing, the time axis is also indicative of ligament strain. The lower strength of the older specimen is apparent. To study this age effect, two kinds of analysis were conducted. First, the average material properties of the six youngest specimens (age 16-26 years) were compared with the strength of the twenty oldest specimens (age 48-86 years). Table 4 shows statistically significant differences in elastic modulus, maximum stress and strain energy to failure and the associated strains. The difference in the strains results primarily from the fact that all of the younger specimens failed by a ligamentous tear while the older specimens failed predominantely by bone avulsion fracture. The force and strain at failure for the older groups are below those expected for ligament-bone units in which the bone is not the weakest link and in which the ligament participates in a major way in the failure process (22,24). The parameters of maximum stress, strain energy and strain to failure in the older group does not, therefore, provide true indications of the material properties for the ligaments. The calculation of elastic modulus, however, is made in the prefailure region of the test and does provide data on ligament behavior. It is worth noting that if the stress-strain curves for the specimen from older humans are extrapolated to the

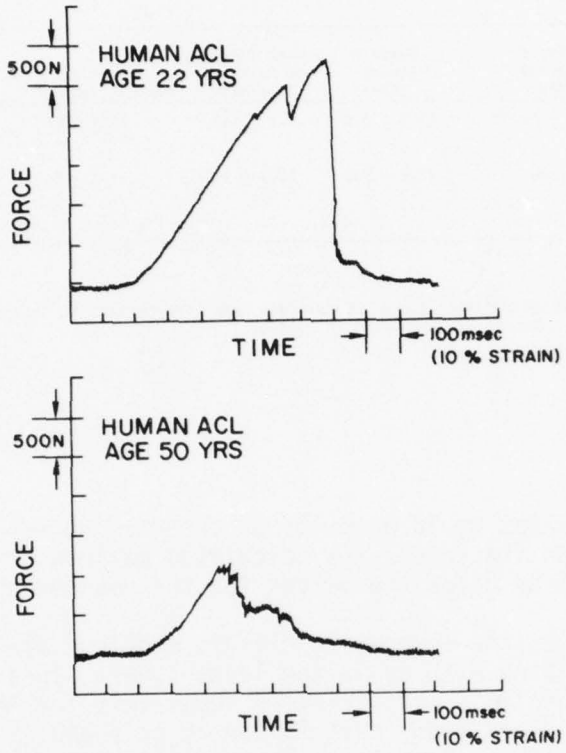

Figure 14. Force-versus-time curves obtained during tensile failure test for two human femur-ACL-tibia units from a young (22 yr male) and older (50 yr female) donors. The older specimens had a lower stiffness and failed at a lower force than the young specimen despite a larger cross-sectional area. Failure mode was bone avulsion for the older specimen and ligamentous for the young specimen.

TABLE 4

COMPARISON OF MATERIAL PROPERTIES[a]

	No. of Specimens	Elastic Modulus (MPa)	Maximum Stress (MPa)	Strain Energy to Failure (N-m/ml)	Strain at Maximum Stress[a]	Strain at Failure
Older Human (48-86 yrs)	20	65.3 ± 24.0[b]	13.3 ± 5.0[d]	3.1 ± 1.5	30 ± 10.0[c]	48.5 ± 11.9[c]
Younger Human (16-26 yrs)	6	111 ± 26	37.8 ± 9.3	10.3 ± 3.1	44.3 ± 8.5	60.25 ± 6.78

[a]Statistical comparison of the Welch modification of student's t test (two-tailed) of each value on each line as compared with that on the line below it are indicated as follows:

[b]$p < 0.01$

[c]$p < 0.001$

strains at which the ligament would normally be expected to fail (in the absence of premature bone-avulsion fracture), the calculated maximum stress and strain energy values would still be below the values for the younger specimens.

In a second analysis, the changes in elastic modulus, maximum stress and strain energy were correlated with age using least square linear regression techniques. The regression analysis was performed separately for those specimens which failed by ligamentous tear and those that failed by bone avulsion fracture as shown in Figures 15a and 15b. Statistically significant decreases with age were found for elastic modulus, maximum stress, and strain energy for specimens that failed by a ligamentous mode. No significant correlation was found for the specimens which failed by avulsion fracture.

The ligament-failure group included all of the specimens obtained as the result of trauma plus the three younger cadaver preparations. In order to exclude possible antemortem effects which might have affected the three cadaver specimens, the regression analysis was repeated using only the eight preparations in which trauma was involved. The result of the analysis was nearly identical with and without inclusion of the three cadaver preparations.

The regression analysis performed for specimens from older humans involved the thirteen amputation specimens and four cadaver preparations. No significant correlations with age were found in this group, and there was no identifiable difference between the mechanical properties for the amputation and cadaver specimens.

It is of interest that the age regression lines for the two failure groups (ligamentous mode and bone-avulsion mode) intersect near 50 years of age, the same

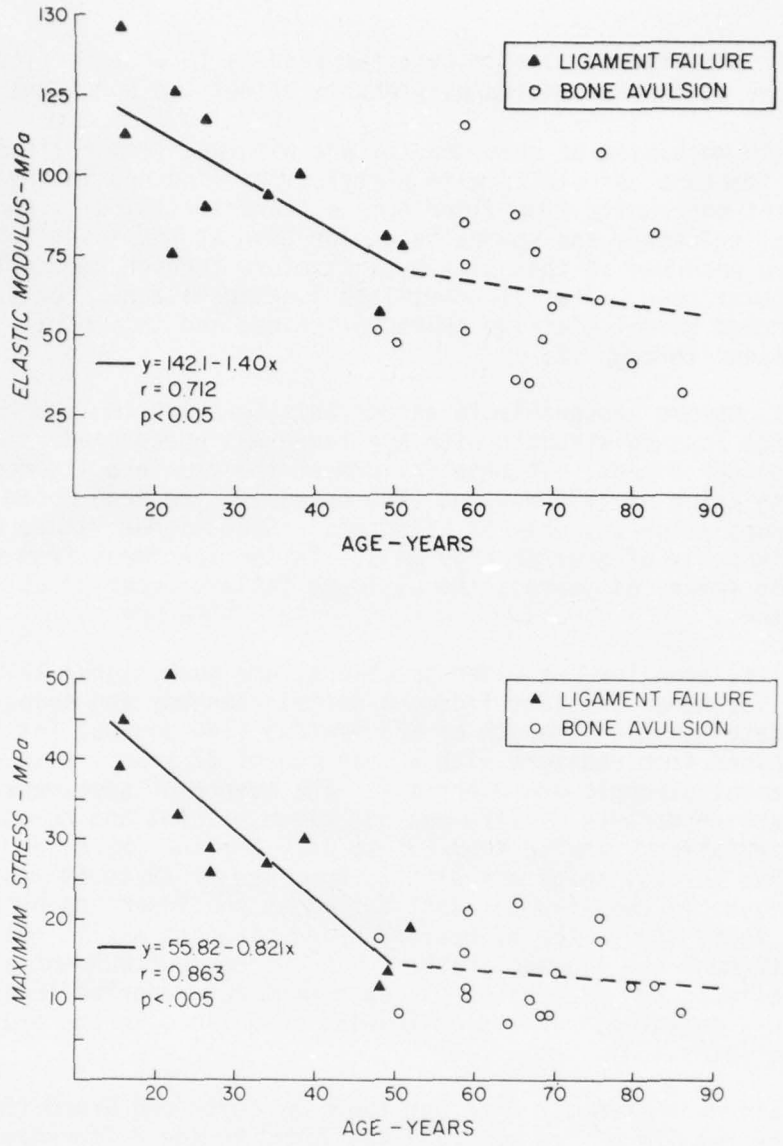

Figure 15a and 15b. Calculated elastic modulus (15a) and maximum stress (15b) for human femur-ACL tibia units correlated against donor age. Elastic modulus was determined in the linear prefailure region of the force-elongation curve. The solid line is a statistically significant correlation for those specimens which failed by ligamentous tear (Δ). No statistically significant correlation with age was noted for specimens that failed by bone avulsion fracture (dashed line;0).

age at which the predominant failure mode changes. The data show that there is a continuous decline in strength and elastic modulus with advancing age and that the process involves both the ligament and bone in the preparation. However, after

the age of 50, it is difficult to attribute the results to an age effect per se because significant antemortem variables probably affect the specimens tested.

The change in mechanism of specimen failure with age from a ligamentous mode to bone-avulsion fracture correlated with histological findings in the failed specimens. The ligament-bone units from older humans characteristically showed a decrease in cortical thickness and spares tabecular bone at the insertion of the ligament. Failure occurred at that site by a fracture through the cortical and underlying trabecular bone. The fibrocartilage junctional zone, for the most part, was intact and showed normal staining characteristics, and only rarely did the fracture plane extend through it.

Although it appears reasonable to expect an alteration in ligament strength with age, the large loss in strength with age found was unexpected. In the specimens from younger humans, ultimate failure of the cruciate ligament unit occurred at a mean value of 1730 Newtons (390 pounds). The mean donor body mass for the younger population was only 53 kilograms. Even higher values would be expected for individuals of greater body mass. In the specimens from older humans, 48 to 86 years old (mean, 64 years), the ultimate failure occurred at an average value of 734 Newtons.

These results, even for the older specimens, are much higher than those previously reported for human cruciate ligament units. Kennedy and associates (19) reported an ultimate tensile strength of 626 Newtons (140 pounds) for isolated human specimens obtained from cadavers with a mean age of 62 years, with no correlation between ligament strength and donor age. The number of specimens tested by Kennedy in the earlier decades of life was not given. Trent and co-workers (33) reported a wide variation, ranging from 285 to 1718 Newtons (mean, 620 Newtons; 140 pounds) in five autopsy specimens with a donor age of 29 to 59 years. They reported that frequently the ligament unit failed at the insertion of the ligament in the bone. In their study, the highest recorded strength was in the one specimen that failed through the ligament rather than the bone attachment site. Potential antemortem effects and the causes of death were not reported in either study, and the testing was performed under slow loading conditions in the study conducted by Trent and associates (33).

The correlation of strength with age found by Noyes and Grood (26) is in disagreement with the results of Kennedy (19) who found no age difference. The regression analysis indicates that with each decade between 16 and 50 years, a decline in ultimate strength of 20.8 percent, of the mean value of twenty years, occurs. A similar decline in strain energy density absorbed prior to failure of 21.0 percent per decade, and a decline in elastic modulus of 12.3 percent per decade was also found. These values are based upon the results from the specimens in which significant antemortem effects could be excluded. After the age of 48 years, we found no significant correlation between strength parameters and age. The older specimens were obtained either from cadavers or from amputations performed for chronic vascular insufficiency. It is reasonable to assume that antemortem factors such as disuse state, disease and ischemia may affect the properties of these specimens. In general, the preparations from older humans represent the type of specimen that is available for experimental studies unless specific steps are taken to obtain other specimens. It is of interest that the strength properties for amputation specimens, obtained as a result of vascular disease do not differ significantly from those for the older cadaver specimens. Although variations in

strength occurred among the preparations from older humans, all of them failed by avulsion of bone beneath the insertion of the ligament.

The difference in the mode of failure (ligament failure in the young versus bone avulsion in the old) deserves special emphasis. Histological analysis of the older human preparation showed a rather marked thinning of the cortical bone immediately beneath the fibrocartilaginous ligament-bone attachment site. The underlying trabecular bone also appeared to have wider spaces between trabeculae, and the individual trabeculae seemed thinner than in the specimens from younger humans.

In a previous study with monkeys, it was reported that the period of eight weeks of immobilization in a cast resulted in a change in the failure mode of the ligament units from ligament failure to bone avulsion (23,24). This was correlated with bone resorptive changes at the sites of insertion of the ligament. It is well known that in humans, loss of bone mass occurs starting in the fourth decade (96). The effect of this phenomenon, particularly in individuals in whome the process is advanced or when it occurs in association with other disuse or disease states in the presence of osteopenia, appears to have important implications to the strength of the osseous components of ligament units.

Studies of age-related changes in the mechanical properties of collagenous soft tissues have shown increases in tensile strength and stiffness and decreases in elongation properties with aging (3,4,11,97,98). These studies involved young animals and the results appear to specifically reflect a maturation process rather than aging. The changes in mechanical properties during maturation are probably related to changes in insoluble collagen, increased intermolecular and intramolecular cross-linking, and increases in the collagen/glycosaminoglycan and collagen/water ratio (97). The physiochemical and mechanical changes that occur in collagen after completion of maturation, which can be ascribed to an aging process, in the absence of adverse environmental or disease factors, cannot be defined at present. Galante (20) studied human collagen fiber preparations from the annulus fibrosus and blamed alterations in load-elongation properties with age on a degenerative process. Tkaczuk (32) studied human lumbar longitudinal ligaments and found significant decreases in strength with age for both degenerated and non-degenerated specimens. Nachemson and Evans (21) reported an age-related five-fold decrease in elastic modulus and failure stress in human ligamentum flavum specimens. This is in agreement with the results presented on the properties of human anterior cruciate ligaments (26).

It is hypothesized that significant reductions in the strength and stiffness properties of ligament units occur with advancing age, much more so than commonly expected. This alteration in functional ligament properties may be a result of many factors, including degenerative processes, disuse effects related to activity status, and superimposed disease states. It may be concluded that results of studies employing specimens obtained from older human donors should be interpreted with caution when applied to the in vivo mechanisms of ligament failure and ligament properties for a younger or healthy population.

TORSIONAL STRENGTH OF THE CADAVERIC KNEE

INTRODUCTION

One of the major mechanisms of knee ligament injury during ejection from a disabled aircraft is excessive rotation of the tibia about its long axis due to large aerodynamic loads. The injury may be either an internal or external rotation injury depending upon the orientation of the foot and leg when it enters the wind stream. Little information is available on the rotational loads and displacements which will produce injury to knee ligaments or the specific patterns of injury which are likely to result (95,96,98-103). To study rotational injuries, two series of tests were conducted. First, the ligaments which restrain internal and external rotations of the tibia were determined. Second, the rotational loads and displacements which correspond to the onset of major failure of the soft tissues were determined. The tests were conducted with the knee flexed to 90 degrees (sitting position) which approximately corresponds to the position when aerodynamic loads are first encountered.

RESTRAINTS TO TIBIAL ROTATION

Methods

Torsion tests were conducted on four cadaveric knees placed in ninety degrees flexion. Skin and muscle were dissected leaving the ligaments and joint capsule intact. The fibula was secured to the tibia with threaded pins. This limits the motion of the distal attachment of the lateral collateral ligament which inserts into the fibular head. Without this fixation the fibula would be hypermobile and it would not be possible to adequately test the function of the lateral collateral ligament. The femur and tibia were potted, using methylmethacrylate bone cement, inside aluminum tubes to assist in obtaining secure fixation during testing. Pins were placed through the tubes, bone cement, and shaft of the tibia to guard against possible failure of the bone cement-tube interface under large torsional loads.

Tests were conducted using an Instron biaxial tension-torsion servo-controlled electrohydraulic test system interfaced to a ModComp II mini-computer. The femur was mounted rigidly in a horizontal position. The knee was flexed to ninety degrees, as in the sitting position, and the vertical tibia was secured to a rotary actuator whose axis was parallel to the axis of the tibia. Care was taken during mounting of the tibia to insure that the rotation axis of the actuator passed between the medial and lateral intercondylar eminences and between the insertion of the anterior and posterior cruciate ligaments. This was verified for each knee at the completion of the test. The anterior-posterior location of the axis ranged from the insertion of the anterior medial band of the ACL to two centimeters posterior of this point. This method of mounting maintains a fixed, rotational axis between tests and eliminates the order of ligament sectioning as an important experimental factor.

The torsion tests were performed with the rotary actuator under angle control. A zero net vertical force between the tibia and femur was maintained with the aid

of the linear actuator operating in load control. While the net force is zero, the testing procedure does permit internal joint contact forces due to tightening of the ligaments and the dissimilar geometry of the femur and tibia.

To determine the restraints to tibial rotation provided by each ligament the following procedure was employed. The tibia was rotated fifteen degrees internally and then externally at the rate of 15^{o}/sec. The restraining torque, produced by the ligaments and the angular displacement of the tibia were monitored and recorded. A ligament was sectioned and the test repeated. A drop in restraining torque results equal to the contribution of the cut ligament. The drop in torque is determined to obtain the contribution of sectioned ligament at 7.5 degrees, 10 degrees, 12.5 degrees and 15 degrees rotation. Finally, the contribution of the ligament, as a percent of total restraining torque in the intact knee is calculated. This process is repeated for each major ligament and capsular structure.

The ligamentous restraints studied were the anterior (ACL) and posterior (PCL) cruciate ligaments, the medial (MCL) and lateral (LCL) collateral ligaments, the iliotibial band (ITB) the popliteus musculo-tendenious unit, and the medial and lateral capsules. The capsules were each subdivided into anterior, mid and posterior thirds. The anatomy as described by Slocum (88) was employed for determining the medial collateral ligament and medial capsule. The lateral capsule was divided as follows; anterior third from the lateral margin of the patella to Gerdy's tubercile, mid third from Gerdy's tubercile to just anterior of the LCL, and posterior third the remainder back to the mid popliteal region. The oblique popliteal ligament, and the ligaments of Humphrey and Wisberg were considered to be capsular structure in this study.

External Rotation

A torque vs. angle curve is shown for one specimen in Figure 16. The relation is non-linear exhibiting increasing rotational stiffness with increasing torque. The lowest stiffness occurs near zero torque which corresponds to the unloaded neutral position of the knee. Hysteresis is evident and results from the viscoelastic properties of the ligament. The origin of the larger hysteresis for external rotation in this specimen is not known. Consistent with our previous findings (12), an increased rate of one order of magnitude did not significantly affect the torque vs. angle relationship.

The primary restraint to fifteen degrees of external tibial rotation (foot pointing away from thebody centerline was the medial collateral ligament (MCL) accounting for $49.7 \pm 5.4^{*}$ percent of the total restraint. None of the other ligaments provided more than fifteen percent of the total as shown in Figure 17. While the mid medial capsule only provided an average of fourteen percent of the total restraint, it accounted for 35.4 percent in one knee with a hypertrophied capsule. Wide variations in the contribution of the mid medial capsule have also been found in studies of the restraints to varus and valgus displacements of the knee (104). While it is felt that most of the variation is due to differences between specimens, the possibility of experimental variations in dissection technique cannot be completely excluded.

*mean ± standard error of the mean.

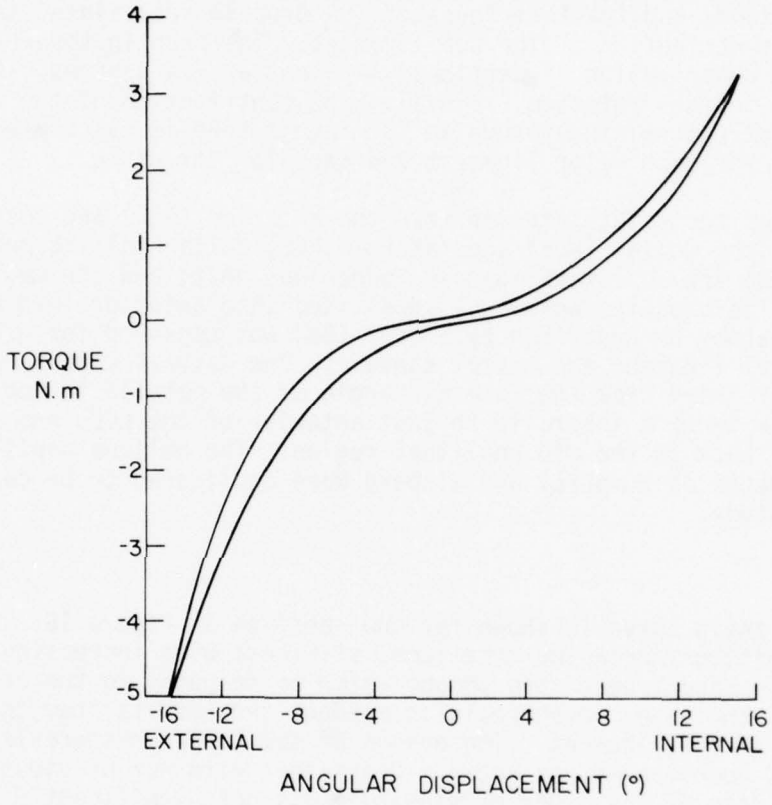

Figure 16. Torque versus angle curve for one knee specimen. Torque is in newton-meters and angle in degrees. Internal rotation cause the foot to point towards the body center. Hysteresis due to the viscoelastic properties of the ligaments is apparent.

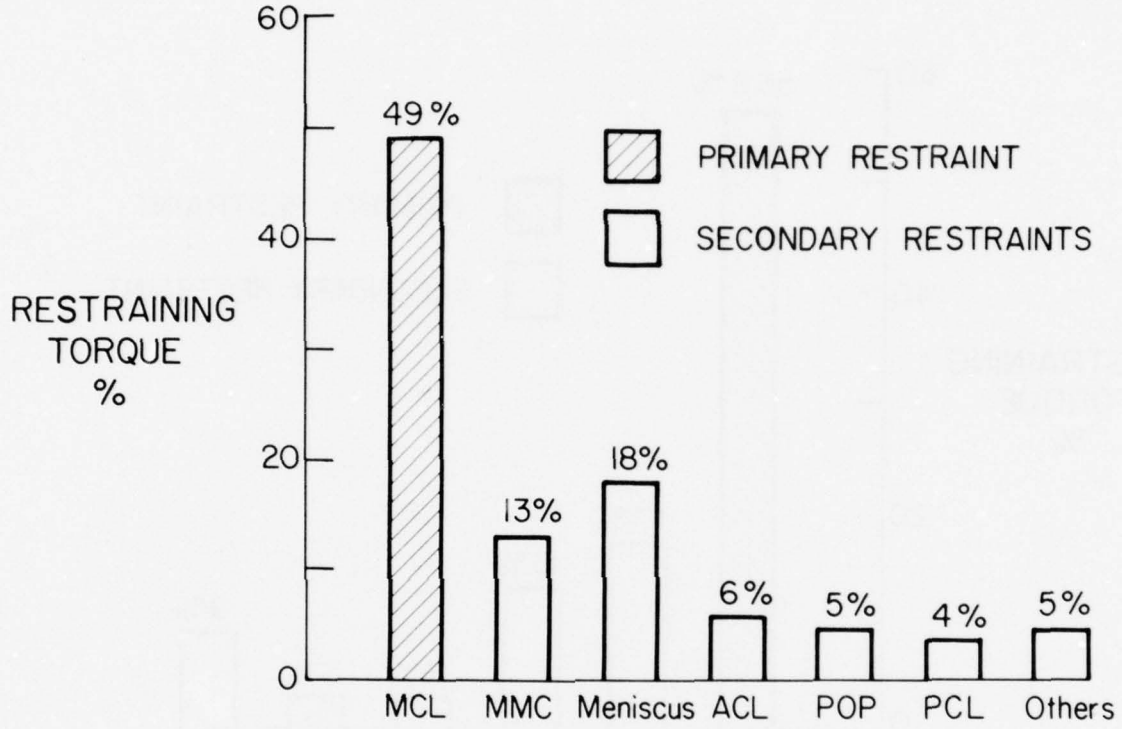

Figure 17. Restraining torque as a percent of total torque due to each ligament for external tibial rotation. The medial collateral ligament (MCL) is the primary restraint. All the other ligaments and capsular structures provide only a secondary restraint.

Internal Rotation

The primary structure which resists internal tibial rotation, shown in Figure 18, was the posterior cruciate ligament which accounted for 48.2 ± 13.2 percent of the total. Important secondary restraints included the iliotibial band

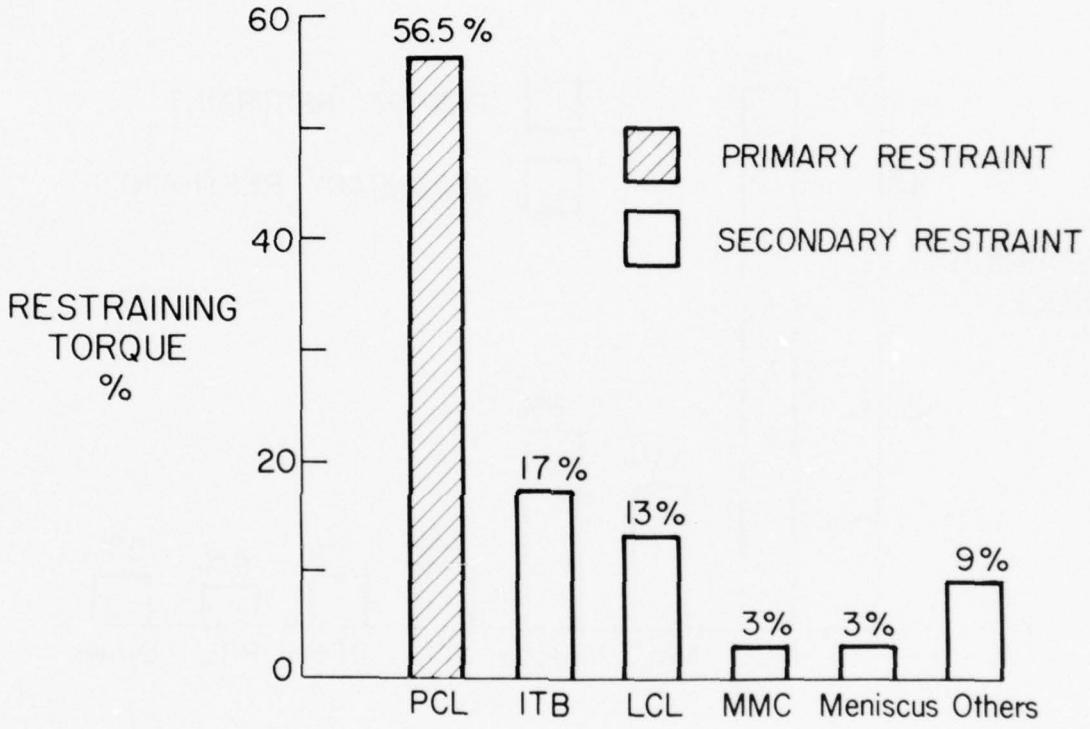

Figure 18. Restraining torque as a percent of the total torque due to each ligament for internal tibial rotation. The posterior cruciate ligament, PCL, is the primary restraint. All the other ligaments provided only secondary restraints.

16.8 ± 6.0 percent and lateral collateral ligament 15.9 ± 8.8 percent. None of the other structures accounted for more than six percent of the total restraining moment.

In conducting these tests it was not possible to fix the iliotibial band as in vivo. This likely results in a low estimate of its passive restraining function.

Additional active restraining moments are also expected in vivo from the popletius musculo-tendon unit even though it has little passive function.

TORSIONAL FAILURE LIMITS

Methods

The onset of failure during the application of excessive internal and external tibial rotations was determined in six cadaveric knees. This was accomplished by applying successively larger angular displacements to each knee, and monitoring the changes which occurred in the torque vs. angle curve from test to test. Two types of changes were found. The first was a relatively small reduction in torque at each angle due to stress relaxation and incomplete recovery between tests. When the period between tests was increased, the magnitude of the reduction was found to decrease.

The second change found was a sudden drop in torque which occurred, similar to the drop in force seen at the linear load point during tensile failure tests of the anterior cruciate ligament. This drop in torque was always associated with a large change in the torque vs angle curve for subsequent tests.

The test sequence used was to first demonstrate the reproducible nature of the torque vs angle curve at an angular displacement well below the point of anticipated failure. A test was then conducted at a larger displacement, followed by a repeat test at a lower displacement to verify the specimen was still intact. The larger displacement test was then repeated as a further check on the integrity of the specimen. If no failure was evident, a larger angular displacement was applied and the verification sequence repeated.

Onset of Failure

Tests were conducted for external and internal rotation on each of six specimens obtained from three male trauma victims. The age, weight and height for each donor is presented in Table 5. The average donor age was thirty-three years

TABLE 5

DONOR CHARACTERISTICS

SPECIMEN NO.	Age Yr,	Height m	Weight Kg.
65,66	27	1.74	86.6
67,68	44	1.75	93.5
69,70	24	1.78	73.0
Average	33.3	1.76	84.4

with a height of 1.76 meters (69.3 inches) and weight of 84.4 kilograms (185 pounds). The data obtained using the specimens from the 49 year donor fell within the range of values obtained on the other four specimens showing no consistant evidence of age effects.

The peak torque reached during successive external rotation tests are shown in Figure 19 for one specimen. The points have been connected by straight lines to

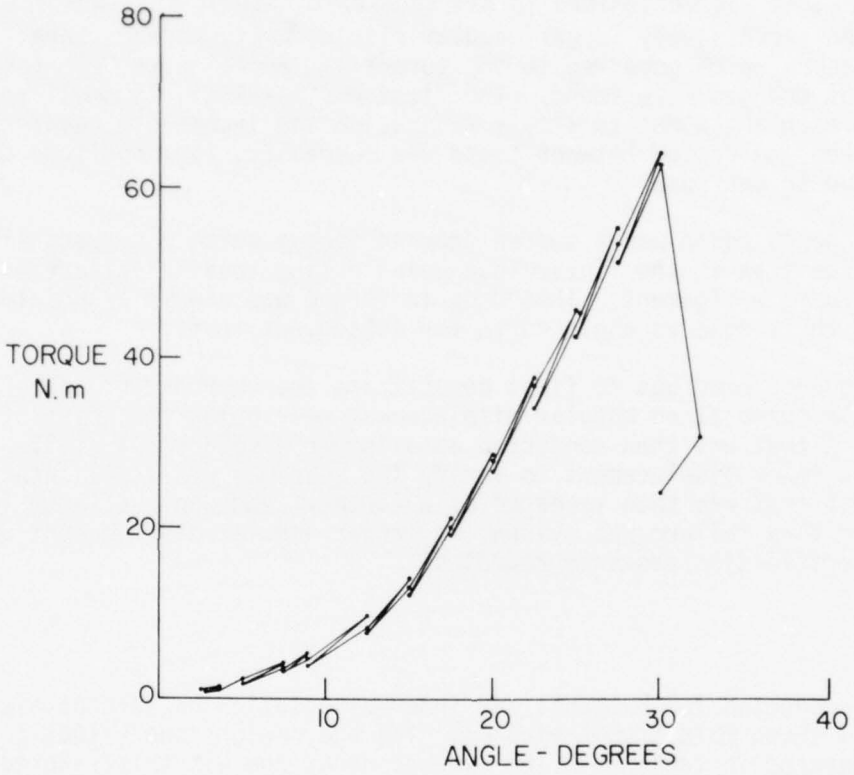

Figure 19. Peak torque versus peak external angular rotation for sequential tests on one specimen. The solid lines connect successive tests. The first test at any angle is followed by a test at a lower angle. The displacement is increased to repeat the original test, and then a test is conducted at a new larger angular displacement starting the sequence over again. The peak torque at each angle decreases with each test due to stress relaxation. The large decrease in torque due to failure that occurred above 30 degrees rotation is apparent.

aid in determining the order of testing. Each angle tested has three points plotted. These correspond to the first test at that displacement, the first repeat after conducting a test at the next smaller displacement, and a second repeat after a test at the next larger displacement. The torque at each angle becomes successively lower

due to stress relaxation. The large drop in torque which accompanies failure at 32.5 degrees is clearly evident. The failure shown occurred during the first test to the next larger displacement. Some failures, however, occurred not during the first test, but during the first repeat test.

The mean angle, torque, and joint stiffness just prior to failure are summarized in Table 6. The average external rotation required to initiate failure was

TABLE 6

TIBIAL TORSION FAILURE LIMITS

	EXTERNAL	INTERNAL
Angle (^{o})	37.5 ± 2.4	30.3 ± 1.45
Stiffness (N·m/^{o})	4.41 ± 0.18	6.00 ± 0.26
Torque (N·m)	66.7 ± 4.8	77.7 ± 7.5

*means ± standard errors (n=6)

37.5 degrees. This is approximately seven degrees higher than the 30.3 degrees of internal rotation required to initiate failure. Although the angular displacement required to initiate failure was larger for external rotation, the torque at failure was less (66.7 Newton meters, external ; 77.7 Newton meters, internal). This is due to the lower stiffness of the knee to external rotation.

DESIGN LIMITS

The establishment of design limits for ejection seats involves two basic steps as presented in the introduction. The first is selection of an acceptable injury level. The second is determination of the mechanical limits which correspond to the injury level in Air Force pilots. These steps will be reviewed in detail here with emphasis upon the limitations of the currently available data and the extrapolations required to satisfy Air Force requirements. While the resulting design limits developed will have a large undertainty associated with them, they will represent a reasonable starting point for the development of protective hardware. Further experimentation and evaluation will be required before these limits can be considered operationally acceptable.

SELECTION OF INJURY LEVEL

The first step is the selection of an acceptable injury level. While the goal of no injury to a crew member is desirable, it fails in practical application. The basic problem is associated with the definition of injury. One of the unique

characteristics of most living tissue is their ability to repair themselves. This is a normal processes which occurs on a continual basis. Its function is to maintain tissue integrity in the presence of the limited life span of individual cells, and the deterioration (fatigue) caused by normal functional forces. Thus, we are led to the conclusion that injury is best considered as a continuum from limited micro trauma at one extreme to massive tissue disruption at the other extreme. The selection of an acceptable injury level must consider both the extent of the injury and the ability of the tissue to heal itself.

The clinical assessment of injury performed by a physician in the treatment of patients is of limited value in setting an injury level for several reasons. First, no studies have been conducted to accurately correlate clinical assessment of injury with the joint load vs. joint displacement data which we must use as our present criteria. Second, clinical assessment is often inaccurate with recent evidence indicating that many significant injuries are overlooked and not properly diagnosed (105). Finally, the serial progressive mechanisms of ligament failure with visual continuity after loss of functional ability indicates that clinical diagnosis cannot adequately assess either the degree of ligament fiber rupture or disruption to the blood supply.

One potential solution to this dilemma is to base the permissible injury level upon biomechanical failure tests. In this approach we assume that if we prevent the ligaments from entering the region of major failure, as determined from their load vs. length curve, that any micro trauma which does occur will be of a limited nature and within the capability of the tissue to repair itself without resulting in a permanent loss of function. This is reasonable so long as we can relate the load vs elongation curve to the degree of disruption of collagen fibers within the ligament. This has been done on a limited basis using scanning electron microscopy as discussed in "Mechanical Properties of Ligaments, Micro-Trauma".

From these studies we propose the linear-load point as the maximum acceptable injury level. The linear load point is shown in Figure 3 for an anterior cruciate ligament-bone prepartions and is designated by a "2". It is believed that the sudden drop in force seen at this point is caused by complete rupture of one or more collagen fiber bundles, and represents a significant local injury which should be avoided. In the anterior cruciate ligament shown in Figure 3, this point occurs significantly prior to maximum force and complete failure. In the medial collateral ligament shown in Figure 6 however, this point is essentially coincident with maximum force and complete failure.

As previously discussed, the linear load point is similar to the onset of yielding in metals, and some ligaments behave in a more "brittle-like" fashion than others. The analogy between linear load and yielding appears reasonable so long as the analogy is based upon occurrence of irreversible failure and not the mechanism involved. One result of considering this analogy is that it highlights the desirability of employing a safety-factor like that used in the engineering design of structures. The magnitude of the safety factor for the biological system, however, need not be as high as that used for structures because of the self healing nature of tissues.

Force vs. Displacement

Given that the injury level is to be based upon the linear load point with a safety factor applied, it must still be decided if the force or the displacement at linear load should be used as the design criteria. This will depend to a large extent upon the protective system employed. Systems which limit the applied force (e.g., windshields) or the allowable displacements (e.g., belts) can be envisioned. In the following discussion we will consider only displacement systems although a force approach could have been considered in a similar manner.

Effects of Age

One of the advantages of using a displacement limit is that the amount of tissue strain to the linear load point is independent of age as seen from Table 4. In contrast, linear stress is a strong function of age and a protective system based upon force levels would have to be designed for the oldest crew member to be protected.

Strain Rate and Disuse Effects

Strain rate and disuse effects are important to consider in evaluation potential injury level criteria to ensure the experimental data does not have a bias based upon either the method employed for testing or the population of test specimens used.

The influence of strain rate on the failure mechanics of ligaments was discussed in "Factors Affecting Mechanical Properties, Strain Rate Effects". The data from seventeen matched pairs of Rhesus anterior cruciate ligaments shown in Table 2 indicates that the strain to linear load is independent of strain rate over the range tested. Note this is not true of either the linear force or the strain to maximum force.

Similar to strain rate, Noyes (24) has found no significant change in the strain to linear load for anterior cruciate ligaments after eight weeks immobilization. This was not true of the linear load, the maximum stress, or the strain to maximum stress. It thus appears that the displacement or strain to linear load may be a reliable quantity which is less likely to be affected by test methods and ante mortem variables than most other parameters.

APPLICATION TO AIR FORCE CREW MEMBER

Now that a potential criteria has been selected for establishing design limits, it is necessary to determine an appropriate value for the population of crew members to be protected. Since it is not possible to conduct tests upon live subjects it is necessary to employ data obtained from cadaveric specimens. The application of such data to the establishment of design limits requires that the potential differences between the population to be protected and the population of test specimens must be carefully evaluated. The factors which can produce a bias in the experimental data may be divided into three major areas; 1) factors which describe differences between the population of crew members and the population of donors at the time of their death (i.e., antemortem factors), 2) factors which relate to changes in the properties of the tissues after death (postmortem factors)

and 3) factors which relate to the methodology used to collect the experimental data.

Antemortem effects of donor age have already been discussed. Anthropometric variables of height and body weight still need to be evaluated. This requires anthropometric data on the target population. Differences in life style, level of activity and physical conditioning between populations may be evaluated by considering the effects of immobility and disuse. This was found not to affect the displacement to the onset of major failure. The final antemortem factor considered here is the cause of death. The specimens listed were all obtained from donors who committed suicide, two by drug overdose, one by gunshot. These causes of death are not thought to affect ligament properties. However, this does not eliminate the possibility that unknown antemortem diseases might have affected the ligaments.

Postmortem effects relate primarily to how soon after death the specimens were obtained along with the method and duration of storage prior to testing. These facts have previously been discussed in "Factors Affecting Mechanical Properties, Effects of Freezing". The available data indicates that alterations in mechanical properties induced by the storage methods we employed are probably small in comparison to specimen-to-specimen variations.

The last category of factors to be considered are those related to testing methodology. Two of the most important are the test rate and the method of mounting. Test rate has been discussed previously and it does not appear to have a strong influence upon the extent of elongation prior to failure. Mounting must be considered because the fixed rotational axis employed during testing is not physiological. The use of a fixed axis was necessary, however, to eliminate the order of ligament sectioning as an important and experimental variable, and to reproduce torque vs. angle characteristics in the failure tests. If the knee specimens were allowed to seek their own axis of rotation, as occurs during flail injuries, an increase in the rotation to failure would be expected. This is because any axis shift away from the actual axis would tend to unload the ligaments under the most tension and most likely to fail. The physical principal which governs the axis shift is the minimization of energy required to produce a given angular displacement. A lower energy input is normally associated with a smaller probability of failure. This does not completely exclude the possibility that an axis shift could cause a redistribution of mechanical energy into tissues less able to absorb it, thereby producing an earlier failure.

Design Limits

As a starting point for the development of a protective system the following design limits are suggested.

Internal Tibial rotation - 17.5 degrees
External Tibial rotation - 20 degrees

These limits are based upon the lowest value for the onset of failure (linear load point) in the torsional test data presented in Table 7. Thelimits include a safety factor of 1.5.

TABLE 7

RANGE OF FAILURE LIMITS

	EXTERNAL		INTERNAL	
	Low	High	Low	High
Angle (°)	30	43	26.5	35
Stiffness (N·m/°)	3.99	5.66	4.99	6.79
Torque (N·m)	54.1	62.2	44.3	88.0

These design limits should only be considered tentative and not used as operational limits until further experimental verification is performed.

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